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Research Article

An Initial Epidemiological Study of Aortic Aneurysms in the Central Australian Indigenous Population

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Introduction

The epidemiology of abdominal aortic aneurysm (AAA) in Caucasian populations is well defined. Studies show that the prevalence of AAA in men aged greater than 65 years is 6-8% with the disease being six times more common in men than women [1]. Data exists regarding the epidemiology of AAA in other ethnic groups, notably Asian [2], Chinese [3], and New Zealand Maoris [4]. In contrast, there is no data regarding the epidemiology of AAA among the Australian Indigenous population.

The risk factors for the development of AAA include male gender, hypertension, smoking, hypercholesterolaemia, family history, social deprivation and geographic isolation [2,3,5,6]. Unfortunately, the Indigenous population carries an excessive proportion of this risk burden, for example, 45% of the Indigenous Australians over the age of 15 years are chronic smokers, compared to 20% of non-Indigenous Australians [7]. These facts may favour the development of AAA in the Indigenous population despite the perception among surgeons that AAA is rare.

Screening for AAA has been demonstrated to be an efficacious and cost effective process in high-risk groups when targeted [8]. Given the potential, but unknown, epidemiology of AAA in Indigenous Australians, this study sought to determine prevalence of AAA in this population group and secondarily determine if a screening program is warranted.

Keywords: Indigenous Health; Aortic Aneurysm; Vascular Surgery; Epidemiology; Screening

Methods

A retrospective study of patients with AAA in Central Australia between July 1998 and December 2008 was completed. Ethics approval was obtained from the Central Australian Human Research Ethics Committee.

The data sources utilised in an attempt to capture all AAA in Central Australia during the study period were: Australian Bureau of Statistics (ABS), Alice Springs Hospital (ASH) medical and surgeon records, South Australian (SA) tertiary hospital databases (Flinders Medical Centre, Royal Adelaide Hospital and The Queen Elizabeth Hospital). Initially patients were identified by searching hospital databases for International Classification of Diseases (ICD-10) codes: 171.3 AAA ruptured and 171.4 AAA without rupture. Hospital records were then manually reviewed to gain further information and cross check.

The ABS 'Annual Cause of Death Report' was reviewed; however this report did not identify AAA as a single diagnosis but under other vascular conditions (dissection and other aneurysms) making the data unusable. A custom in-depth search was requested and approved by the ABS. This attracted a $400 fee and the authors were informed that all demographic and ethnic data would be removed if a small number of cases were identified.

The Territory Coroners Office, Department of Births Deaths and Marriages, and the National Coroner's Information System (NCIS) were also approached for data on deaths associated with AAA, however the authors were informed that it was impossible to search databases by diagnosis except in the case of the NCIS, where all demographic and ethnicity data would be removed rendering it unusable for the purposes of an epidemiological study.

Results

The catchment area of the ASH encompasses an area of approximately 1,175,000 square kilometres with a population of 50,260. Indigenous Australians accounted for 47.7% of the population.

ASH records revealed 11 patients with AAA in Central Australia. Of these patients, six were male, and the average age was 74.5 years (age range 53-86 years). None of these patients were Indigenous. Three patients died as a result of AAA rupture. The average documented AAA size was 62mm.

Interrogation of the SA hospital databases revealed eight patients treated with AAA from Alice Springs and surrounds. None of these patients were Indigenous.

The in-depth search of the ABS revealed a total of 18 patients with AAA, 11 of who were male. No other epidemiological data was made available to the researcher; specifically no data regarding the ethnicity of any of these patients was revealed.

Despite reasonable requests for information to multiple government agencies listed, no further data was provided.

Discussion

This study identified a maximum of 37 patients with AAA in Central Australia between July 1998 and December 2008. It must be noted that this is the maximum number and may not be a true representation of the data. No agency was able to provide enough detail to enable the researchers to check if patients were included in both the ASH/ABS numbers and ASH/SA numbers. Thus the true number of identified cases could be as low as 18. This is far lower than one would expect, even amongst Caucasian population of approximately 26,000, given a prevalence of 6-8% [1]. Interestingly no cases of AAA were identified as being Indigenous and whilst it is possible that Indigenous Australians are at low risk of developing AAA despite their multiple risk factors, several possible factors must be considered. Firstly, Indigenous Australians have a significantly reduced life expectancy. An Indigenous male in the Northern Territory has a life expectancy of 61.5 years, below the age when AAA prevalence rises in the Caucasian population. Secondly, given the apparent infrequency of AAA, local medical practitioners may also under-diagnose the condition in this population. Thirdly, the high prevalence of diabetes mellitus among the Indigenous population might offer some protection, given the negative correlation between diabetes and development of AAA [9]. Finally, major barriers were encountered in the collection of the data for this study for both Indigenous and Caucasian patients, highlighting the poor quality of some databases, a cumbersome bureaucracy, and the difficulties in conducting retrospective studies.

What we do know is that Indigenous Australians remain at high risk of cardiovascular disease, disproportionate to non-Indigenous Australians [10]. Furthermore those in remote communities have particularly poor access to health care. In addressing the aim of this study, the retrospective approach used to determine epidemiology of AAA among the Indigenous population has proved unsatisfactory. In future, efforts to define the epidemiology of AAA in this population would best be made prospectively by screening a targeted sample of the population with ultrasonography, which is non-invasive, quick and ethically acceptable. This will allow the researchers full control of the data and opportunities to explore other environmental as well as potentially important genetic factors.

Conclusions

The epidemiology of AAA amongst Indigenous Australians remains unknown. There exist many barriers to the conduct of retrospective epidemiological study of disease among Indigenous Australians. Thus prospective investigator driven studies would best way to overcome these obstacles.

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